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Cervical Spine Functional Anatomy and the Biomechanics of Injury Due to Compressive Loading

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Objective: To provide a foundation of knowledge concerning the functional anatomy, kinematic response, and mechanisms involved in axial-compression cervical spine injury as they relate to sport injury.

Data Sources: We conducted literature searches through the *Index Medicus*, SPORT Discus, and PubMed databases and the Library of Congress from 1975–2003 using the key phrases *cervical spine injury, biomechanics of cervical spine, football spinal injuries, kinematics of the cervical spine,* and *axial load.*

Data Synthesis: Research on normal kinematics and minor and major injury mechanisms to the cervical spine reveals the complex nature of movement in this segment. The movement into a single plane is not the product of equal and summative movement between and among all cervical vertebrae. Instead, individual vertebrae may experience a reversal of motion while traveling through a single plane of movement. Furthermore, vertebral movement in 1 plane often requires contributed movement in 1 or 2 other planes. Injury mechanisms are even more complex. The reaction of the cervical spine to an axial-load impact has been investigated using cadaver specimens and demonstrates a buckling effect. Impact location and head orientation affect the degree and level of resultant injury.

Conclusions/Recommendations: As with any joint of the body, our understanding of the mechanisms of cervical spine injury will ultimately serve to reduce their occurrence and increase the likelihood of recognition and immediate care. However, the cervical spine is unique in its normal kinematics compared with joints of the extremities. Injury biomechanics in the cervical spine are complex, and much can still be learned about mechanisms of the cervical spine injury specific to sports.

Key Words: catastrophic injury, whiplash, injury mechanisms, spinal cord, axial load

Because of the potentially catastrophic and life-altering nature of cervical spine injury (CSI), much concern exists regarding the prehospital management of the cervical spine–injured athlete. This is evidenced by a multiprofessional task force effort initiated by the National Athletic Trainers' Association to establish general guidelines for the acute care of the spine-injured athlete.[1] Major CSIs, although rare compared with sprain and strain injuries to the extremities, are troubling because of mortality rates and the potential permanent loss of neural function. A CSI requires an immediate and deliberate, yet sensitive, response. The highest rate of severe neck injuries has occurred in American football and rugby.[2–8] Other sports and activities that contribute to a high rate of CSI are wrestling, diving, recreational diving, ice hockey, gymnastics, and horseback riding.[3,5,9]

The more severe CSIs associated with athletics can be attributed to compressive forces from axial loading.[10,11] Clinically, a major CSI results in compromised integrity of the cervical segment due to fracture, dislocation, subluxation, or ligamentous tearing, leaving the cervical spine unstable. White et al[12] defined clinical instability in the spine as more than a 3.5-mm horizontal displacement of one cervical segment on another. Obviously, the athletic trainer is unable to detect the presence of such a diminutive irregularity in the structure of the spine and must, therefore, assume the worst-case scenario.

Motion in one plane at the cervical spine requires the contribution of complementary motion from individual vertebrae in other planes.[13–15] This further complicates the kinematics of the cervical segment and the resultant injury mechanisms. Considering the mechanism of injury is an important first step for the on-field assessment of any athletic injury. An athlete with a significant spinal cord injury may not immediately present with emergent signs and symptoms. Therefore, understanding the kinematics of the cervical spine is important for the athletic trainer, not only in helping to appreciate the following sections regarding injury mechanisms but also in allowing for a more effective evaluative tool after CSI.

The purpose of this literature review is to provide a foundation of knowledge concerning the functional anatomy, kinematic response, and primary mechanisms involved in CSI during participation in sports, specifically as they relate to axial-compression forces. A secondary purpose of this review is to demonstrate the need for research investigating sport injury mechanisms of the cervical spine.

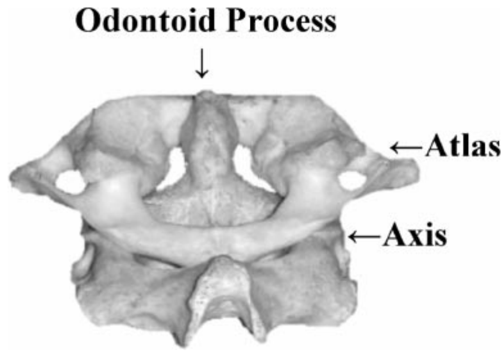

Figure 1. Posterior view of C1 (atlas) and C2 (axis).

FUNCTIONAL ANATOMY

The cervical spine's range of motion is approximately 80° to 90° of flexion, 70° of extension, 20° to 45° of lateral flexion, and up to 90° of rotation to both sides.[16] However, movement in the cervical spine is complex, because pure uniplanar movement does not accurately portray the motion between cervical levels, and movement into any range is not the simple sum of equal motion from one vertebra to the next.[13]

Normal Kinematics of the Upper Cervical Spine

The first cervical vertebra, the atlas, has often been labeled the cradle, because its articulation with the occiput of the skull provides a cradle for supporting the head (Figure 1).[15] The atlas articulates with the occipital condyles, and its primary motions are flexion and extension. Normal flexion to hyperextension at the atlanto-occipital joint ranges from approximately 15° to 20°.[15,16] Rotation and lateral flexion between the occiput and atlas are not possible due to the depth of the atlantal sockets, in which the occipital condyles rest. Rotation to one side causes the contralateral occipital condyle to contact the anterior wall of its atlantal socket and the ipsilateral condyle to contact the posterior wall of its respective atlantal socket.[15] Similarly, lateral flexion requires the contralateral occipital condyle to lift out of its socket, a movement that is restrained by the tight atlanto-occipital joint capsule.[15]

The weight of the head is transferred to the cervical spine through the lateral atlanto-axial articulations of C2, the axis (see Figure 1). The superiorly directed odontoid process extending from its body rests within a facet on the atlas that is created by the anterior arch and allows the atlas and head to rotate from side to side as one unit. The normal ranges of rotation of C1 on C2 are reported to be 50° to each side.[16] However, results have varied, and this range has been noted to be 32° in cadavers,[17] 75.2° through radiographic techniques,[18] and 43° using computed tomography scanning.[19] Nevertheless, this rotational ability of the atlanto-axial joint is possible due to the stabilizing function of 3 primary ligaments (transverse, alar, and apical), which act to hold the dens as a ''fixed post'' on which the atlas can rotate.[20]

Rotation is also possible at C1 through C2 because, unlike the atlanto-occipital joint, the lateral superior and inferior articulating facets of the atlas and axis create a biconcave surface. The concavity of each articulating surface is due to the articular cartilage of the inferior and superior facets and is not visible on a radiograph.[15] This characteristic allows for the anterior and posterior translation of the articular surfaces, and as the atlas continues to rotate, it settles into the axis as the superior articular process on each side slides down the anterior and posterior rims of the convex inferior surfaces. The biconvex nature of the atlanto-axial articulation means that cervical spine flexion and extension often create motion in the direction opposite that being experienced in the atlas.[13–15] Thus, when the cervical spine is flexing, the atlas extends, and when the cervical spine extends, the atlas flexes. This coupling motion is possible because the atlas is balanced on the concavity of the axis, and when the line of compression moves anterior to this balance point, as when the neck is extended, the atlas moves into flexion. The reverse follows as the cervical spine flexes, moving the line of compression posterior to the balance point and creating extension at the atlas. This coupling, or reversal of motion, is a unique characteristic of the spine, may be experienced at different levels, and will also be important in understanding mechanisms of injury (Figure 2).

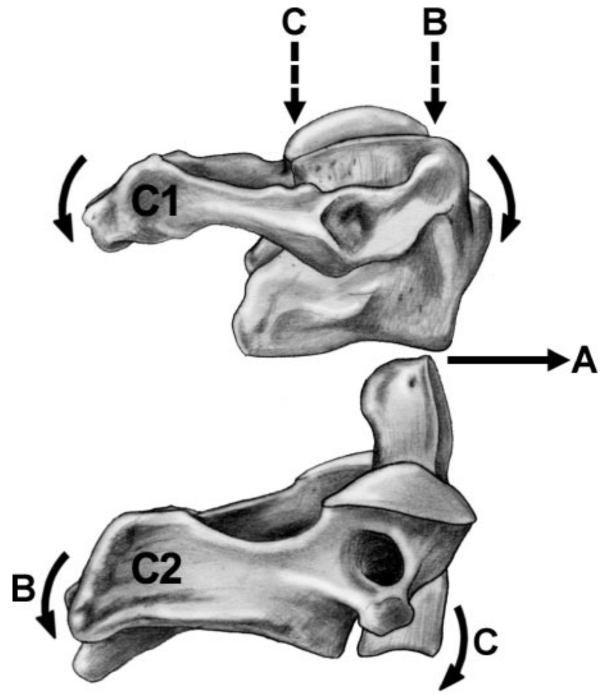

Figure 2. The biconvex nature of C1 and C2. A, Translation. B, Extension of C1 creating flexion in C2. C, Flexion of C1 creating extension in C2.

Another feature of the atlanto-axial joint also found in other segments in the cervical region is that pure rotation of the atlas on the axis does not occur without a small degree of extension and lateral flexion and sometimes flexion.[18] Again, the line of vertical forces being distributed through the occiput to the atlas as the head moves determines the amount of coupling motion in the atlas as it balances between the head and the axis.

Normal Kinematics of the Cervical Column

At the C2 through C3 junction, the upper cervical spine meets the remaining, more typical cervical column. The body of the axis acts as a ''root'' within C3, securing the upper cervical spine in the remaining cervical column.[15] The articulating surfaces of the inferior and superior intervertebral joints are similar to a saddle joint, maintaining anterior-posterior and medially and laterally directed concavities.[21] This orientation of the cervical bodies of the mid to lower cervical column allows for rotation and flexion movements but is re-

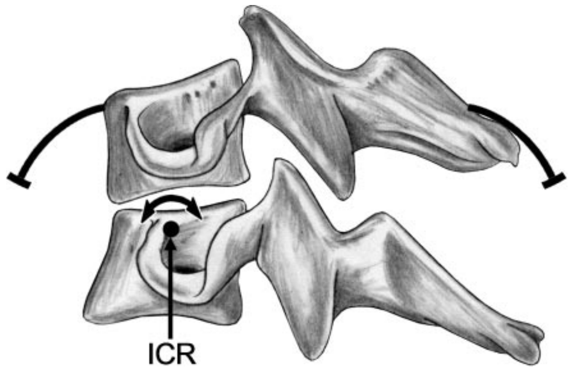

Figure 3. Instantaneous center of rotation (ICR).

sistant to lateral flexion. Lateral flexion is possible as a combined movement in the cervical column but only due to coupled rotational movement in each segment to that side.[21]

General flexion and extension motion of the neck does not necessarily reflect the movement among vertebrae in the cervical spine. In fact, a vertebra may experience its greatest range of motion in flexion or extension before the cervical column itself has fully flexed or extended. Furthermore, a vertebra may experience a large range of movement in one direction while the cervical column on the whole exhibits movement in the opposite direction.[13,14] The order of contribution from cervical segments into flexion and extension varies by level as well. Through high-speed cineradiography, Van Mameren et al[13] determined that flexion is initiated at the lower cervical spine (C4 through C7), followed by motion at C0 (occiput) through C2, C2 through C3, and then C3 through C4. The C6 through C7 segment undergoes a brief reversal of motion into extension, followed by a reversal of motion at C0 through C2. The C6 through C7 segment contributes to the end ranges of flexion. Extension is also initiated in the lower cervical spine (C4 through C7) and is followed by the beginning of motion at C0 through C2. The middle range consists of varied movement from the mid cervical region, whereas the lower cervical spine is the last to contribute as the column moves into terminal extension.[22]

This unique motion characteristic of the cervical vertebrae is explained by Penning[14] and Amevo et al[23] through a concept known as the instantaneous center of rotation (ICR). The center of rotation for a particular vertebra is actually located near the superior aspect of the inferior vertebral body (Figure 3) and can be used to explain the reversal of motion observed in the cervical spine. The ''reversal of curve'' that occurs at a vertebra is due to its role as a pivot point in the cervical column. As the lines of force are transmitted down the cervical column, the vertebrae experience flexion or extension depending on the location of the force vector relative to the ICR. Hence, if the cervical column is moving into flexion, but the force vector passes behind a specific vertebra's ICR, then that vertebra will extend. Penning[14] used the ICR theory to describe the biomechanics of injury in the cervical column. The reversal-of-curve phenomenon in the cervical spinal segment is further elucidated through the work of Nightingale et al[24–27] in analyzing injury to the cervical spine during ''buckling'' and is discussed later.

BIOMECHANICS OF INJURY

In the early literature of CSI, researchers focused on the movement of the head during injury and ascribed the primary mechanism of suspected injury in the cervical spine to that specific movement.[28–30] However, further study has shown that the observed motion at the head during injury is not a reliable indicator of spine movement responsible for creating the injury.[24,25,31–33] The biomechanics in the spine and extent of injury to the spine depend on the impact location on the head and the orientation of the cervical spine at the time of impact.[24–27] The initial, and often the more critical, injury occurs as soon as 2 to 30 milliseconds after impact, well before observed motion in the cervical spine and head occurs.[24–26]

Axial Loading in Football

The mechanism for injury that has received the most attention in athletics is axial loading. Only 13% of the 209 football-related injuries that resulted in permanent cervical quadriplegia between 1971 and 1975 resulted from hyperflexion (10%) and hyperextension (3%), whereas 52% were attributed to axial loading.[34] According to the National Center for Catastrophic Sport Injury Research, a total of 107 cases of permanent cord injuries in football occurred between 1977 and 1989, with most resulting from tackling.[10] More recently, 15 cases of quadriplegia were reported in high school football between 1991 and 1993, with the principal cause attributed to axial compressive loading.[35]

Axial loading occurs when the head and neck are flexed to approximately 30°, as in a head-first tackle. In this position, the normal lordotic curve disappears, which removes the energy-absorbing elastic component of the region. When contact is applied to the crown of the head or helmet in the football player, the cervical spine experiences a compressive load from the torso. As the padding provided by the helmet reaches its absorptive limits, the head then reverses direction, resulting in an increased compressive load as the cervical spine is compressed between the head and torso. When this compressive force exceeds the spine's absorption capabilities, soft and hard tissue components fail. Compressive load limits of the cervical vertebrae have been calculated at 3340 to 4450 N.[25–27] Interestingly, the upper compressive load limits of the cervical spine have been reached in less than 11 milliseconds during simulated impacts using a speed and mass equivalent to approximately half those of a conditioned athlete.[36] Moreover, CSI occurs when the compressive loads on the cervical spine are increasing at an increasing rate, demonstrating that during an axial load, the potential for serious neck injury exists independent of neck strength.[36]

Too frequently, these injuries are brought about by a conscious effort to spear, or use the crown of the head as the initial point of contact during a tackle. Despite rule changes, equipment improvements, and an overall decrease in the number of catastrophic injuries that result from these significant epidemiologic findings, spearing appears to be as prevalent now as it was before.[37–40]

Cervical Spine Buckling

During axial loading in the cervical spine, compressive forces result in a transient deformation, or buckling effect.[25] This buckling produces large angulations within the cervical spine as a means of releasing the additional strain energy that has been produced from the vertical loading and is the causative factor of injury (Figure 4).[24,25,27]

Nightingale et al[24,25,27] assessed the dynamic responses of

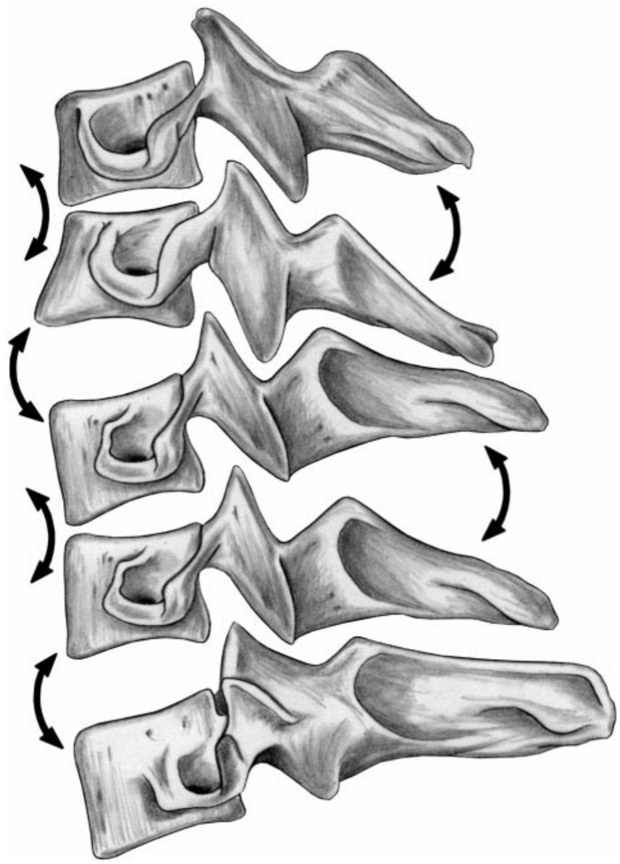

Figure 4. Buckling effect in the cervical column under axial load.

the cervical spine to axial loading using high-speed video and cadaver specimens mounted (inverted) to a drop-track apparatus. Buckling occurred in 1 of 2 distinct orders.[24–26] First-order buckling resulted in extension of the upper cervical spine through C5 and flexion through T1. Second-order buckling created flexion of C1 through C3, extension in C4 and C5, and flexion in C6 through T1. The levels at which the spine reverses its motion are where pivot points have been created, as in the abrupt reversal of curve described earlier.[14] Buckling and injury were produced in the specimens within 2 to 31 milliseconds after impact, well before observable movement in the head and neck complex was noted (20 to 100 milliseconds).[25,26] In essence, a cervical vertebra can almost immediately be placed in an extreme position of hyperflexion or hyperextension during buckling, even though the head and neck complex is not yet experiencing observable motion. This fact is critical for the athletic trainer to understand: motion at the head is not a reliable indicator of the motion at various levels in the spine; therefore, the potential for injury should not be underestimated.

Although investigating human injury mechanisms with cadavers is valuable, a limitation lies in the inability to account for the contribution of forces and joint stability from soft tissues. Even though injury occurs before the approximate time of reflexive muscular contraction (60 milliseconds),[41] some degree of muscle activity and stiffness can be assumed to be present in the athlete's cervical column at the moment of impact. Furthermore, to our knowledge, most of the research performed on vertical and axial-loading biomechanics have involved load applied to a cadaver specimen in a vertical direction.[25,26,42–46] Few instances in sport place the athlete in a pure upright or inverted position when receiving an axial load. More often, the player purposely lowers the head or falls, and the head and trunk travel in a parallel or oblique direction relative to the ground. The effect of gravity, coupled with the static stabilization of posterior muscles in the upper back and cervical region in these situations, could affect the buckling mode and direction of resultant force vectors. Researchers to date have not attempted to simulate these internal and external forces in a nonvertical position and their effect on impending injury, and they have not assessed the effect of adding mass and size to the head, such as when wearing a helmet. Further research in this area specific to the functional demands and injury mechanisms in sports is necessary.

Head Orientation and Padded Surfaces

Nightingale et al[24–26] assessed dynamic response in cadavers to axial impacts by adjusting the contact-surface orientation so the head received impact anterior to the vertex of the head (15° and 30°), at the vertex (0° or midpoint), or posterior to the vertex of the head (15°). The greatest extent of injury was when the impact was at the vertex or anterior to the vertex of the head, with no injuries to the cervical spine with an impact posterior to the vertex.[25,26] For the anterior and vertex impact conditions, only 2 of 6 specimens did not experience injury, whereas 2 specimens experienced injuries at multiple levels.[26] The authors correlated injury risk to impact location. Specifically, when the head experienced an impact directly at or anterior to the vertex of the head, the neck-loading vector responsible for moving the head out of the path of the torso was too small and could not accelerate the head out of the way before injury occurs. However, the further the impact point is in the posterior direction, the easier it is for the cervical spine to flex and move the head out of the way of the increased force of the torso.[26]

Nightingale et al[24–26] also investigated impact into padded surfaces. Padded surfaces within helmets are used globally in sport and act as protective covering for the extremities and objects such as goalposts. Yet the relationship of this padding to the kinematics of the cervical spine has not been established. Investigators, in fact, have failed to demonstrate that padding acts to protect the cervical spine, and it has even been shown to *increase* the likelihood of major injury to the cervical spine.[24–26]

Using the same methods of cadaver preparation and impact platform orientation, the researchers covered the rigid surface with a padded material.[25,26] Impact forces at the head decreased, as would be expected, but the resultant neck forces increased, causing more severe injuries than with nonpadded impacts, regardless of impact orientation.[25,26] The greater impulse, due to the padding, allows for impact forces to be experienced over a greater time, and in most scenarios in sport, this is desirable. However, with regard to the cervical spine, the longer the head is placed in axial compression, the longer the time injurious forces are applied to the neck. Therefore, the padding creates a ''pocket'' for the head during impact. This pocketing causes the head to become depressed into the padded foam surface, which acts to ''hold on'' to the head.[26] Hence, a pocketing event increases risk of injury to the cervical spine due to the increased time of exposure to compressive force.[25–27]

Impacts to the head in contact sports are often into a surface with some degree of padding. This can include padding over equipment, a pad worn by an opponent, the soft tissue of an opponent, compressive playing surfaces, or padding within the helmet itself. These situations may all present some degree of pocketing, making it much more difficult for the spine to escape an axial load. Although the presence of padding provides a risk for CSI, its use is necessary to protect against the more commonly occurring blunt trauma encountered in sport that involves the skull or other anatomical structures. Athletic trainers should consider the type of impact surface involved in a suspected CSI and, according to the findings of research, should appreciate the potential severity of injury due to pocketing and impact orientation.

Whiplash

Although more common in motor vehicle crashes,[47] whiplash injury can also take place in contact sports.[32] An example of a whiplash-type injury in football occurs when the quarterback is sacked from behind, particularly if the quarterback is not expecting to be hit. Yet, contrary to what is generally considered to be an injury caused by extreme range of motion in the head or neck,[32] some cervical injuries from whiplash are due to compressive forces.[48–50] The difference in the mechanism between whiplash and the typical axial load is that the compressive force is superiorly directed from below the cervical spine,[49] as opposed to an inferiorly directed force from axial load.

The combined efforts of several researchers using human subjects,[51] cadaver specimens,[43–45,48,52] and mathematical modeling[53] have provided a detailed timing of events in the cervical spine during whiplash to explain the compressive mechanism. The first joints and segments of the body to experience movement during a whiplash are the hips, back, and trunk.[51] These structures of the body not only move forward after a rear-end collision but upward as well. This upward thrust of the trunk compresses the cervical spine.[48,50,51] Coupled with the forward displacement of the trunk, this combination of motions causes the head to revolve backward into extension, creating tension and buckling where the lower cervical segment extends and the upper cervical segment flexes.[51] Peak extension of the neck during whiplash is reported to be 45°, which is not beyond the neck's normal physiologic limits and, hence, is not the actual cause of injury.[48,49,54] Rather, the injurious mechanisms (compression, forward trunk displacement, and buckling) act together to abnormally rotate individual vertebrae into extension. As a vertebra experiences this rotation, the anterior components are separated and the posterior components, particularly the facet joints, experience extreme compression.[48,50,51] Minor fractures of the inferior facet joints have been discovered in cadaver specimens[50] and during autopsy in cadavers after motor vehicle crashes.[55] Therefore, injury in the vertebral region is not necessarily due to shearing or excessive cervical spine hyperextension. Rather, an individual vertebra positioned between the flexed and extended regions of a buckling spine extends around an abnormally high axis of rotation, which forces it into the superior facet joints of the subjacent vertebra (Figure 5).[48–50,52,54,56,57]

In describing the events that occur during whiplash, most researchers have studied subjects sitting in car seats with headrests.[48,49,51,52,58] However, whiplash-type injury in sport other than competitive auto racing is more likely to occur when the athlete is in an upright position without a head restraint. The upward thrust of the torso may not be as great in an upright position as in a seated position, possibly minimizing the buckling and compressive effect. However, abnormal rotations in the vertebrae may still take place, particularly because there is no posterior restraint to the head and neck.

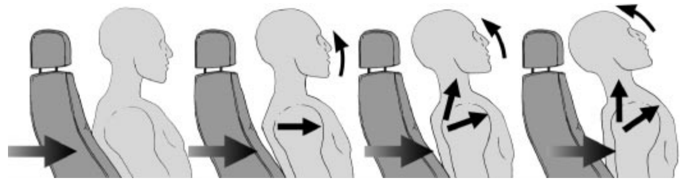

Figure 5. Forward displacement of the trunk and compression and upward rotation of the cervical spine and head.

If initial radiographs do not reveal cervical fractures after whiplash, typically the diagnosis is generalized to soft tissue injury, and no further diagnostic techniques are pursued.[54] However, threats to cervical column integrity are still possible from whiplash and may not be clearly identified with any radiographic technique, which may lead to an underestimation of recovery time. Research investigating the true mechanisms and resultant injury of whiplash specific to sport is warranted.

Pressure Gradient Injuries

Spine injury and permanent cord damage can still occur even if the osseous and soft tissues remain intact.[59] Imagine the dural sac as a water-filled balloon. As the balloon is bent or squeezed at the midsection, the diameter of the balloon at the site of the squeezing diminishes as water inside the balloon flows away from the site of increased pressure. As the water collects on either side of the restricted area, the balloon distends as increased water volume applies greater stress to the walls of the balloon. Normal cervical motions apply standard pressure changes to the spinal cord that do not result in injury. However, when the neck is subjected to extreme ranges of motion at high speeds or in the presence of cervical spinal canal stenosis, excessive pressure may be applied to neural tissues. If this stress exceeds the resiliency of the neural tissue, these structures may be compromised, much like excessive or rapid squeezing of a water balloon may cause the balloon to rupture.

Pressure experienced by the cord and changes in the geometry of the spinal canal under axial load have been investigated by Chang et al.[59] Specifically, these researchers measured diameter changes in the spinal canal and cervical spine segment height during axial-induced injury in 10 cadaver specimens. The authors prepared the cadaver cervical spine segments with the spinal cord removed and placed a tube pumped with water in the spinal canal to measure the occlusion created during impact. A 5.95-kg weight was then dropped vertically from a height of 1.53 m. In 8 of 10 specimens, the tube in the spinal canal was completely occluded at the moment of injury. Additionally, the specimens underwent an average decrease in spinal segment height of 8.9 mm at the point of impact, only to recover 35% of their original height.[59]

Clearly, at the moment of axial-load impact, the cervical spine and spinal canal undergo major transient geometric changes. Injuries to the spinal cord are often described as cord contusions and are likely a direct result of the decrease in canal diameter. After this injury, the neural tissue responds like any tissue in the body to a concussive force, which can result in neurapraxia or spinal shock. Therefore, cord injury may be present even with negative radiologic findings and a stable spine segment. Careful and detailed neurologic evaluation should be performed after spinal shock to ensure complete resolution of the initial inflammatory phase.

CONCLUSIONS

As is the case with every joint of the body, our understanding of the mechanisms of CSI will ultimately serve to reduce their occurrence and increase the likelihood of immediate recognition and appropriate care. However, the cervical spine is unique in its normal kinematics. Injury biomechanics in the cervical spine are complex, and much can still be learned through continued research on the reaction of the cervical spine to injury mechanisms specific to sports.

Furthermore, athletes who place themselves in positions known to be associated with spinal cord injury run a higher risk of spinal cord injury and paralysis. Athletic trainers must not only understand mechanisms of injury to the point of being able to recognize them on the field but also be able to demonstrate that, oftentimes, it is an athlete's decision to place himself or herself in a compromising position that leads to injury.

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